



Review on Epidemiology of Lumpy Skin Disease

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Abstract

Lumpy Skin Disease (LSD) has caused significant financial losses in the livestock industry. It is attributed to the Lumpy Skin Disease Virus (LSDV), a member of the Poxviridae family, with the Neethling strain as the original form. LSDV belongs to the Capri poxvirus genus, which also includes the sheep pox virus and goat pox virus. LSD is a contagious disease in cattle, known for causing nodules to form on the skin. While it rarely results in death, it has a high rate of illness, particularly among calves. The disease affects only cattle and water buffalo, leading to decreased milk and beef production, as well as instances of abortions in females and infertility in males. The origins of LSD can be traced back to Zambia in 1929, and it is considered to be prevalent in Africa. However, it spread beyond Africa in 1984, reaching Madagascar and several countries in the Arab Gulf Peninsula and Middle East. Recently, LSD has been reported in countries that were previously free from the disease, such as Jordan, Syria, Lebanon, Turkey, Iran, and Iraq, posing potential economic losses for the livestock industry. This review aims to discuss the current status of LSD and its spread to previously unaffected countries, highlighting concerns about its impact on these regions.

Introduction

Lumpy Skin Disease (LSD), also known as Pseudo urticarial, Neethling Virus Disease, Exanthema Nodularis Bovis, or Knopvelsiekte, is a highly impactful transboundary viral disease that is emerging in the cattle population. It affects cattle of all ages and breeds and is caused by a virus belonging to the family Poxviridae, genus Capri poxvirus, and species LSDV. This virus is closely related to poxviruses found in sheep and goats [1]. The disease is characterized by the presence of fever, nodules on the skin, mucous membranes, and internal organs, as well as high morbidity, low mortality, emaciation, enlarged lymph nodes, edema of the leg and brisket, mastitis, orchitis, and in some cases, death [2]. LSD was first identified in Zambia in 1929 and has since spread to other African countries. It is currently prevalent in most African nations and has also expanded into the Middle East region [3].

According to a study by [4], there has been a widespread outbreak of LSD in various regions of Ethiopia, with the disease now affecting almost all regions and agro-ecological zones of the country. LSD is considered one of the most economically significant livestock diseases in Ethiopia, typically occurring at the end of summer and beginning of autumn. The transmission of LSDV is primarily attributed to blood-feeding arthropods such as hard ticks, biting flies, and mosquitoes [5]. Diagnosis of LSD is typically based on clinical signs, epidemiology, histopathology, virus isolation, and PCR [6].

The disease has substantial economic impacts, including reduced milk production, temporary or permanent sterility, deaths, beef loss, loss of draft animal power, abortion, loss of condition, and damage to the hide. While there is no antibiotic treatment for LSD, supportive care may be available. Major strategies for controlling and preventing LSD include ring vac-



cination, quarantine, movement restrictions, and control of insect vectors [3].

In Ethiopia, there has been minimal research conducted on this disease, with only a few studies reporting on risk factors, epidemiology, seroprevalence, and financial impacts [7]. Additionally, there was an outbreak of this disease in my region last summer, which affected numerous cattle and resulted in the deaths of many due to a lack of understanding of its epidemiological aspects and the misconception of it being a simple skin wound. Therefore, the purpose of this seminar paper is to conduct a comprehensive review of: Epidemiological features of lumpy skin disease on a national and international level. Financial implications of lumpy skin disease at the level of individual farms and on a country-wide basis.

Literature review

Definition: Lumpy Skin Disease (LSD) is a viral illness in cattle that can lead to a range of symptoms, from mild to severe, such as fever, skin nodules, mucous membrane and internal organ nodules, leg and brisket swelling, lymph node inflammation, and in some cases, fatality. The disease has significant economic consequences, including reduced milk production, loss of traction power, weight loss, stunted growth, abortion, infertility, and skin damage, resulting in substantial financial impact [1].

Etiology

Lumpy Skin Disease (LSD) is caused by the Lumpy Skin Disease virus (LSDV), a member of the Poxviridae family, Chordopoxvirinae subfamily, and capripoxvirus genus, with the prototype strain being the Neethling Virus. LSDV is a pleomorphic, enveloped virus with a brick- or oval-shaped double-stranded DNA structure, measuring 350*300nm and having a molecular weight of 73 to 91 kilo Daltons (K Da). The LSDV genome sequence ranges from 145 to 152, and its terminal genomic sequences contain a unique complement of at least 34 genes that are responsible for viral virulence, host range, and/or immune evasion of the host [8].

All Capri poxviruses have a slow growth rate in cell cultures and may need to be passed through several times to proliferate. They can be cultured on different types of cells from cows and sheep, which leads to easily identifiable changes in the cells. Additionally, the virus can be grown in the chorioallantoic membranes of developing chicken embryos, causing visible pock lesions. The LSDV replicates within the cytoplasm of the host cell, forming distinct eosinophilic inclusion bodies [9].

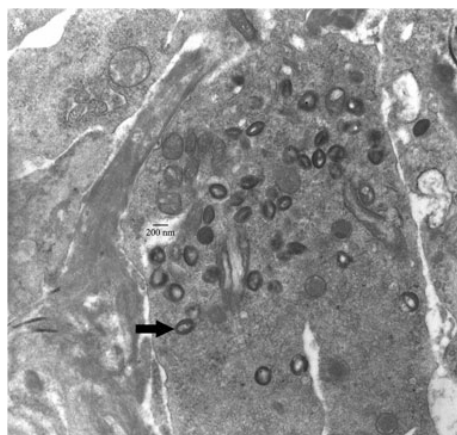


Figure 1: The image shows Capri poxvirus particles extracted from the skin of a goat infected with Capri poxvirus. An arrow points to a specific poxvirus particle [10].

LSDV is sensitive to sunlight and detergents that contain lipid solvents such as ether (20%), chloroform, formalin (1%), and phenol (2%). The virus can be deactivated by heating at 55°C for one hour [11]. However, it is resistant to drying and pH changes, unless the pH is extremely high or low, and can remain viable for months in dark environments such as the skin shed from an infected animal. LSDV can persist in skin plugs for approximately 42 days [12].

Epidemiology

Lumpy skin disease is a significant and economically damaging notifiable illness that causes a decrease in cattle production due to widespread discomfort and long-term weakness [3]. A comprehensive understanding of the epidemiological factors related to LSD, including the pathogen, host, and environment, could be beneficial for developing control and prevention strategies. It is important to focus on how hosts are exposed to the pathogen in environments that facilitate the transmission and spread of the disease. LSD is more common during the wet summer and autumn months, especially in low-lying areas and along waterways [1].

Geographic distribution

LSD, which originated in Zambia in 1929, has spread to both the north and south over the past seventy years. It is now endemic in almost all African countries, as well as Madagascar, occurring in various ecological zones. However, it has not been reported in Libya, Algeria, Morocco, and Tunisia.

Outbreaks have also been reported outside of Africa, including in the Middle East in 2006 and 2007, in Mauritius in 2008 and in Israel [13]. The epidemiological trend of LSD indicates that it is currently endemic in most African countries and is spreading further into North Africa, Middle East countries, and Mediterranean regions due to global trade movement in animals and animal products [3,6].

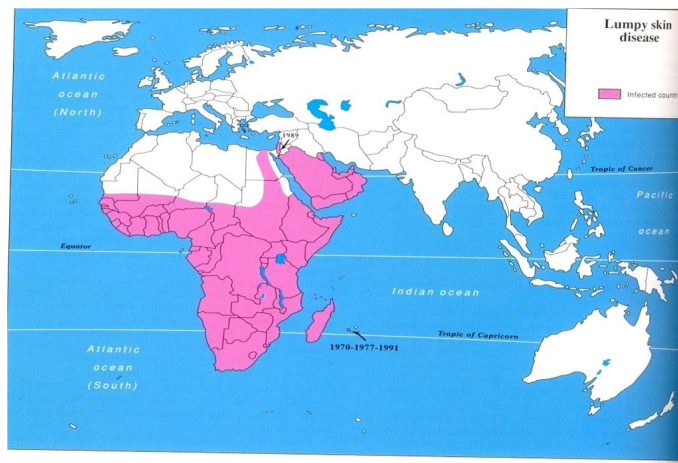


Figure 2: Geographical distribution of LSD, Source: [11].

Species of animal affected

Lumpy Skin Disease (LSD) primarily affects all types of cattle, especially those of European breeds with thin skin, making them particularly susceptible [13]. CapriPoxViruses (CaPVs) are highly specific to their hosts, with only a few exceptions known. There is limited information available on the susceptibility of wild ruminants to LSD. Instances of capripox disease have been observed in domestic Asian water buffalo and Arabian Oryx, but it is unclear whether these animals were infected with LSDV or other related poxviruses such as sheep pox or goat poxvirus [14,6].

While natural cases of LSD have not been observed in impalas and giraffes, it has been demonstrated in both species after experimental inoculation with LSDV. The absence of a reservoir host for the LSD virus suggests that infection may persist in endemic areas at a low level, possibly in unapparent or mild forms within the cattle population [15].

Source of infection

Animals that are showing clinical signs of illness are the primary means of spreading infection to other healthy animals. Nevertheless, the LSD virus can be found in bodily fluids such as blood, skin lesions, saliva, nasal discharge, tears, milk, and semen, and very rarely, in drinking water, which could serve as potential sources for transmission [12,16,17].

Mechanism of Transmission

Direct transmission

The direct spread of LSD can happen when animals use the same feeding and drinking areas, leading to contamination by nasal and salivary discharges from infected animals or ingestion of contaminated food [11]. Calves that suckle may also become infected through contaminated milk. Research has shown that LSDV can be transmitted through semen [18].

A more recent study found that the live virus responsible for LSD can persist in bovine semen for up to 42 days after infection, and viral DNA was still detectable up to 159 days post-infection [16]. During the natural outbreak of LSD in Egypt in 2006-2007, it was discovered that 25% of cows had infected ovaries due to LSDV, and 93% of cows experienced ovarian inactivity and did not show signs of estrus [9]. There is a belief that the virus may also be present in vaginal secretions. It is generally thought that transmission of the virus through direct contact is not very efficient, and field evidence has suggested that the disease is not easily spread from one animal to another [19].

Role of vectors

The spread of LSDV is facilitated by blood-feeding arthropods such as hard ticks, biting flies, and mosquitoes, as reported in studies by [5,7,20]. This type of transmission is considered to be mechanical rather than biological, meaning that the virus is transmitted via contaminated mouthparts of the vectors without actual replication of the virus in the arthropod cells or tissues. Unlike biological transmission, where infectious organisms multiply within the vector, the mechanical mode of transmission does not involve long-term survival or multiplication of the virus in the vectors. Research by [5] demonstrated that the virus can persist for 2-6 days after feeding from infected cattle and can be transferred to susceptible cattle by female mosquitoes, specifically *Aedes aegypti*, during experimental infection.

Recent research has presented new findings suggesting that hard ticks may be involved in spreading LSDV. The study demonstrated that *Boophilus decoloratus* can transmit the LSD virus through different life stages and generations, while *Repicephalus appendiculatus* and *Ambyloma hebraeum* can mechanically transfer the virus [3]. Additionally, mosquitoes such as female *Aedes aegypti* and *Culex quinquefasciatus*, along with other arthropod vectors like tabanids, biting midges, and *Glossina* species, are significant in the transmission of the virus. Non-biting flies such as houseflies, bush flies, and blowflies are also commonly associated with transferring the virus through contact with infected secretions. Furthermore, vermin, predators, and wild birds may act as mechanical carriers of the virus [21].

Epidemiological evidence indicates that LSD outbreaks are closely linked to high populations of insect vectors and the onset of rainy seasons. The spread of the disease is associated with rainy seasons, river basins, ponds where cattle graze, and humid environments that are conducive to insect proliferation [1].

Risk Factors

Host risk factors

Lumpy skin disease affects cattle and causes various ailments. While all breeds and age groups can be affected, *Bos taurus* is more prone to developing clinical symptoms compared to zebu cattle and *Bos indicus* [2]. Among *Bos taurus*, high-producing dairy Channel Island breeds with fine skin are particularly susceptible to LSDV [9]. Infected lactating cows often experience a significant reduction in milk production due to high fever from the viral infection and subsequent bacterial mastitis [3].

In contrast, indigenous breeds such as zebu and zebu hybrids may possess some inherent resistance to the virus [4]. The genetic factors influencing disease severity are not yet fully understood. Factors such as high ambient temperatures, farming practices, and high milk-yielding cows could contribute to the severity of the disease in Holstein-Friesian cattle [22].

Young animals are severely affected by LSDV, and clinical symptoms manifest rapidly [10]. Traditional calf management practices that separate calves from the herd may reduce their exposure risk to the source of infection. Calves in endemic areas may acquire some protective passive immunity from their mothers. Animals that have recently recovered from an LSDV infection are immune for approximately three months [4].

In local zebu cattle, male animals have a higher cumulative incidence of LSDV compared to females, possibly due to stress factors such as exhaustion and fatigue rather than biological reasons. Male animals are often used as draft oxen for heavy labor, making them more susceptible to infection. Additionally, draft oxen may be more vulnerable to biting flies when harnessed in a yoke, and the skin abrasions they sustain during plowing could attract flies capable of transmitting LSDV [4]. The severity of the disease generally depends on the susceptibility, immunological status, and age of the host population, as well as the dose and route of virus inoculation [14].

Pathogen risk factors

Lumpy Skin Disease Virus (LSDV) is a type of capripoxvirus that is resilient to various chemical and physical agents [23]. Capripoxviruses have envelopes containing lipids and are susceptible to detergents with lipid solvents such as ether (20%), chloroform, formalin (1%), phenol, and sunlight. They are also sensitive to sunlight but can endure well in cold temperatures. LSDV is vulnerable to temperatures of 55 °C for two hours, 65 °C for 30 minutes, as well as alkaline or acidic pH. There is no significant reduction in titer when held at pH 6.6-8.6 for five days at 37 °C.

The LSD virus is found in nasal, lachrymal, and pharyngeal secretions, semen, milk, and blood. However, the virus can persist in saliva for up to 11 days, in semen for 22 days, in necrotic tissue at the site of a skin lesion for 33 days, and for six months on fomites such as clothing and equipment. There is no evidence that the virus can survive for more than four days in insect vectors. There is no evidence of the virus persisting in the meat of infected animals, but it may be isolated from milk in the early

stages of fever [10].

Capripoxviruses are highly resilient in the environment and can remain viable for extended periods on or off the animal host. They may persist for up to six months in a suitable environment, such as shaded animal pens. The virus can be recovered from skin nodules kept at -80 °C for 10 years and infected tissue culture fluid stored at 4°C for six months [21].

Environmental risk factors

Environmental factors play a significant role in the spread of lumpy skin disease. They have a major impact on the virus, the animals affected, and the insects that act as carriers, as well as the interactions between them. These contributing factors play a crucial role in the persistence of insect vectors and the transmission of the virus to susceptible animals. Factors such as animals sharing communal grazing areas and watering points, unregulated movement of cattle across borders due to trade and pastoralism, rainfall and wet climates that promote insect breeding, and other reasons for cattle movement from one place to another are potential risk factors for lumpy skin disease [3].

Lumpy skin disease is linked to an increase in the number of insects that act as mechanical carriers [20]. It is more prevalent during the wet and warm conditions of summer and autumn, particularly in low-lying agricultural areas and along watercourses [1]. The warm and humid climate in midland and lowland agricultural areas has been identified as a more favorable environment for the presence of large populations of biting flies compared to the cooler temperatures in the highlands [6].

Morbidity and mortality

The disease is most prevalent during wet and warm weather and decreases during the dry season, according to the [15]. The morbidity rate during outbreaks varies widely, ranging from 3% to 85%, depending on the hosts' immune status and the abundance of mechanical arthropod vectors [14,6]. However, in natural outbreaks, it can reach as high as 100%, with a mortality rate that rarely exceeds 5% but may occasionally reach 40% [10,16].

Pathogenesis

Lumpy Skin Disease (LSD) is caused by the entry of the infectious LSDV through the skin or gastrointestinal mucosa, leading to viremia and a febrile reaction. The virus then reaches and causes swelling of the regional lymph nodes [4]. The mechanism by which the virus causes skin lesions is attributed to its replication in specific cells, such as the endothelial cells of lymphatic and blood vessel walls, resulting in the development of inflammatory nodules on the skin [24].

LSD is a generalized and epitheliotropic disease that triggers localized and systemic reactions, leading to vasculitis and lymphadenitis, which in turn result in edema and necrosis. In severe cases, thrombosis and other symptoms may also be observed [2].

The characteristic skin nodules of LSD may initially exude serum but eventually develop an inverted grayish-pink conical zone of necrosis. Enlarged lymph nodes and secondary bacterial infections are common within the necrotic cores. Multiple virus-encoded factors are produced during infection, influencing pathogenesis and disease [6]. The incubation period of LSD can vary under field and experimental conditions, ranging from

4 to 14 days in experimentally inoculated animals and up to 24 weeks in naturally infected animals [1].

Clinical sign

Lumpy skin disease can manifest in various ways, including acute, subacute, and chronic courses. The virus can cause a range of clinical symptoms, from mild to severe, and animals that develop the disease may experience a two-phase febrile reaction. The main observable clinical signs include a fever ranging from 40-41.5°C, which may persist for 6-72 hours, along with increased tear production, heightened nasal and throat secretions, reduced appetite, decreased milk production, some signs of depression and reluctance to move, skin nodules, as well as swelling of superficial lymph nodes. Nodular lesions can have a diameter of 1-7 cm and appear as round, firm, intradermal, and circumscribed areas with erect hair [1,3].



Figure 3: Cows that are affected by LSD infection exhibit numerous skin nodules.

In more serious instances, sores may form in the mucous membranes of the mouth, trachea, larynx, and esophagus [2]. The dead tissue at the center of these sores can detach from the surrounding skin, creating what are known as sit-fasts. This condition can be made worse by secondary bacterial infections and infestations of fly larvae [14]. Additionally, lesions in the skin, subcutaneous tissue, and muscles of the limbs, along with severe skin inflammation resulting from secondary infections, significantly impair mobility, as reported by [23].



Figure 4: Inverted conical zone' of necrosis and so called a sitfasts lesion.

Pneumonia is a frequently occurring bacterial complication and often results in fatality. Prolonged fever can lead to the absence of the estrus cycle, painful genitalia preventing bulls from mating, and frequent abortion in the early stages [25,21].

Skin nodules are most commonly found on the head, neck, perineum, genitalia, limbs, and udder. They affect the skin, cutaneous tissues, and sometimes the underlying muscle. The severity of clinical symptoms depends on the strain of Capripoxvirus and the breed of the affected cattle. Additionally, in cases of experimental infection, the route of transmission and the virus dosage also play a determining role [1].

Diagnosis

According to [1], the diagnosis of LSD involves considering its epidemiology, clinical signs, necropsy findings, and laboratory tests. Clinically, it can be identified by the characteristic nodular lesions on the skin, which may be present as raised areas of hair, as well as nodules around the nostrils, mouth, vulva, and prepuce. These nodules can either persist as hard lumps or become moist, necrotic, and sloughed [4]. Additionally, edema of the leg and swelling of the superficial lymph nodes are common signs [3].

At necropsy, the disease can be diagnosed by observing nodules on the skin and mucous membranes, as well as swelling of the superficial lymph nodes and other systemic symptoms [14].

Rapid laboratory testing is crucial for confirming LSD. Diagnosis can be made through transmission electron microscopy, isolation and identification of the virus, serological tests, routine histopathological examination, and immunohistological staining [1]. The virus can be isolated from collected biopsies or post-mortem samples within the first week of clinical signs, before neutralizing antibodies develop [1,14]. However, the growth of such viruses is slow and requires several passages in primary cell cultures.

Serological tests are useful for retrospective confirmation but are time-consuming and may have limited presence of detectable antibodies in serum [24]. Real-time PCR is considered the most appropriate technique for diagnosing LSD due to its high sensitivity and good specificity [1,3].

Histopathological findings

Histopathological findings of LSD are highly distinctive and serve as a foundation for diagnosis. The nature of the lesions varies significantly depending on the stage of the disease. During the acute phase, the disease is primarily characterized by vasculitis, thrombosis, infarction, and perivascular fibroplasia. Inflammatory cells, including macrophages, lymphocytes, and eosinophils, infiltrate the affected areas. Additionally, intracytoplasmic eosinophilic inclusions may be observed in keratinocytes, macrophages, endothelial cells, and pericytes. The epidermis and dermis of the infected animal display edema and infiltration with large epithelioid macrophage-like cells.

Similar oedema and infiltration of the epidermis and dermis with large epithelioid macrophage-like cells have been well-documented in cases of sheep pox. These cells are accompanied by plasma cells and lymphocytes in early lesions, while fibroblasts and polymorphonuclear leucocytes, along with some red cells, predominate in older lesions. Endothelial proliferation is evident in the blood vessels of the dermis and subcutis, with lymphocytic cuffing of the blood vessels leading to thrombosis and necrosis. Distinct intracytoplasmic inclusions may be

present in various epithelial elements, sebaceous glands, and follicular epithelium. These inclusions are largely eosinophilic-purple and seem to have a clear halo surrounding them, likely due to processing artifacts. The lesions are generally consistent throughout the body.

Pathological Lesion

Gross lesions

During postmortem examination, nodules can be detected in the subcutaneous tissue, muscle fascia, and muscles. These nodules appear grey-pink with caseous necrotic cores, as well as signs of congestion, hemorrhage, and edema. The subcutaneous tissue is infiltrated with red, watery fluid. Similar nodules may be distributed throughout various organs including the nasopharynx, trachea, bronchi, lungs, rumen, abomasum, renal cortex, testicles, and uterus [21]. Bronchopneumonia may be evident and superficial lymph nodes are often enlarged. In severe cases, there may be synovitis and tendosynovitis with fibrin present in the synovial fluid [14].

Microscopic lesion

Histological examinations reveal characteristic eosinophilic pox inclusion bodies within the cells of epithelial tissues, hair follicles, muscles, and skin glands during the initial phase of skin lesions [14,21]. Additionally, significant lesions of vasculitic necrosis containing cell debris and extensive infiltration of inflammatory cells, predominantly neutrophils, are observed in both the superficial and deep layers of the dermis [4].

Differential diagnosis

Lumpy skin disease can be suspected if clinical signs include prolonged fever exceeding 105.8°F, widespread skin nodules, enlarged lymph nodes, conjunctivitis, keratitis, corneal opacity, and edema in the brisket and legs [2]. Histopathology is a valuable tool for ruling out viral, bacterial, or fungal causes of nodular development in clinical cases. The presence of characteristic eosinophilic intracytoplasmic inclusion bodies in cases of lumpy skin disease is well-documented [13].

According to [21,1], the following are differential diagnoses for lumpy skin disease:

Bovine herpes mammillitis: Lesions are superficial and primarily affect cooler body parts such as teats and muzzle, with no generalized disease.

Hypodermal bovis: Parasitic fly larvae migrate to the dorsal skin of the back, causing nodules with a small central hole through which the larva exits the body, resulting in significant hide damage.

Photosensitization: Dry, flaky, inflamed areas are limited to unpigmented skin.

Ringworm (dermatophytosis): Cattle lesions are grayish, raised, plaque-like, and often itchy. The organism can be demonstrated with a silver stain.

Streptotrichosis (Dermatophilosis): Lesions are superficial, often moist, and appear as crusts or 0.5- to 2-cm diameter accumulations of keratinized material. Common in the skin of the neck, axillary region, inguinal region, and perineum. The organism can be demonstrated by Giemsa staining.

Treatment

Lumpy skin disease is caused by a virus and there is no specific cure for the disease. However, as you mentioned, supportive treatments such as antibiotics to treat secondary bacterial infections, anti-inflammatory drugs to reduce fever and inflammation, and vitamin shots to improve the animal's overall health and appetite can be used to manage the symptoms and support the affected animals during the course of the disease.

It's important to note that while these supportive treatments can help manage the clinical signs and improve the animal's well-being, they do not directly target the virus itself. Prevention through vaccination and biosecurity measures is crucial for controlling lumpy skin disease in affected populations.

Control and Prevention

In endemic areas

In countries like Ethiopia, the control and prevention of Lumpy Skin Disease (LSD) primarily rely on the annual vaccination of cattle above six months of age. Calves born to immunized cows benefit from passive immunity, which lasts for approximately six months [14]. The vaccination approach is commonly used in most parts of the country and often involves ring vaccination around local outbreak foci when they occur [4].

To control LSD, four live attenuated strains of capripoxvirus are currently employed as vaccines. These include the Kenyan sheep- and goat-pox strain (KS-1), the Yugoslavian RM 65 sheep-pox strain, the Romanian sheep-pox strain, and the South African neethling LSDV strain. In Africa, two different vaccines have been widely and effectively used for preventing LSD in cattle populations. The Neethling strain of LSD, which has undergone extensive passages in tissue cultures of lamb kidney cells and embryonated eggs, is commonly used in southern Africa.

In Kenya, a strain of sheep and goat pox virus was passaged 16 times in pre-pubertal lamb testes or fetal muscle cell cultures [1,13]. Due to antigenic homology and cross-protection between sheep pox, goat pox, and LSD viruses, any of these viruses can be utilized as a vaccine strain to safeguard cattle against LSDV [14]. Animals that have recovered from natural infection or have been vaccinated with one of the strains acquire lifelong protection and resistance to infection with any other strain, without becoming carriers [21]. Protective immunity typically develops 10 to 21 days post-vaccination and necessitates an annual booster dose [1].

New areas

The risks of introducing Lumpy Skin Disease (LSD) into new areas include the potential introduction of infected animals, animal products, and contaminated materials [16]. If LSD is confirmed in new areas, it is essential to implement quarantine measures to contain the disease before it spreads extensively. This involves slaughtering infected and in-contact animals, as well as cleaning and disinfecting any equipment that may have come into contact with the disease [21].

Proper disposal of infected animals and their products is necessary to eliminate the source of infection. Additionally, implementing quarantine and movement controls for animals, products, and potentially infected items is crucial to prevent the spread of the disease. Controlling insect vectors, such as using insect repellents, providing insect-proof housing for animals, and applying insecticides, is important to minimize the

mechanical transmission of the virus. Tracing and surveillance activities are also essential to determine the source and extent of infection. Furthermore, ring vaccination is a key strategy for controlling and preventing LSD [21,1].

Status of lumpy skin disease in Ethiopia

In Ethiopia, Lumpy Skin Disease (LSD) was initially detected in 1983 in the western region near the southwest of Lake Tana [4]. Following its initial appearance, a rapid and widespread epidemic emerged, spreading from the northern to the central and southern parts of the country. The national disease report indicated that LSD had virtually disseminated to all regions and various agro-climatic zones in Ethiopia. Due to the extensive spread of the disease and the size and composition of the cattle population in the country, LSD is considered one of the most economically significant livestock diseases in Ethiopia [26,27].

A recent study conducted across diverse agro-ecological zones in Ethiopia revealed an overall observed prevalence of LSD at 8.1% with a mortality rate of 2.12%. The estimated case fatality is 2% [26]. The highest frequency of LSD outbreaks in the country has been documented between September and December, with the peak occurring in October and November, marking the conclusion of the primary rainy season in most parts of the midland and highland agro-ecological zones. Conversely, the lowest number of cases is reported in May [28]. The indigenous local zebu cattle breed known as Fogera, located in northwest Ethiopia, has been reported to exhibit severe clinical manifestations during LSD epizootic occurrences [4,15]. A study in Ethiopia also indicates that communal grazing, watering points, and movement of infected livestock have been linked to the occurrence of LSD [7].

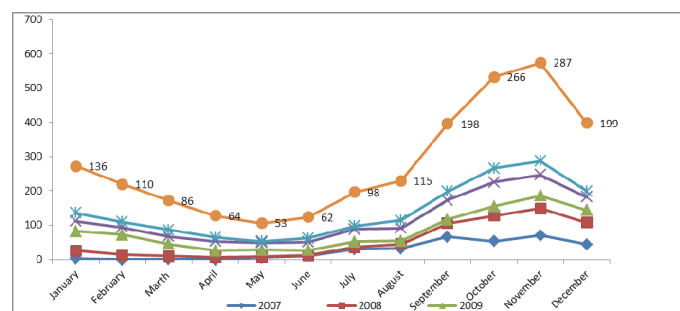


Figure 5: The incidence and timing of lumpy skin disease outbreaks in Ethiopia from 2007 to 2011. Source: [28].

[26] stated that Lumpy Skin Disease (LSD) is among the diseases in Ethiopia that should be promptly reported to the national veterinary services in the event of an outbreak. [28] conducted an analysis of historical data from January 2007 to December 2011, revealing that LSD has been reported in all regions of Ethiopia except Harari and Dire Dawa. The majority of outbreaks are frequently observed in the midland agro-climatic zones of Oromia, Amhara, and the Southern Nations, Nationalities, and Peoples Region. These areas are known to be conducive for the breeding of blood-feeding insect vectors of LSD and have the highest livestock population density in Ethiopia [26].

The darkest shaded areas on the map indicate the highest number of outbreaks, while the lightest shaded areas represent the lowest number of outbreaks [28].

In Ethiopia, there has been limited research conducted on Lumpy Skin Disease (LSD) thus far, with only a few studies addressing risk factors assessments, epidemiological aspects, seroprevalence, and financial impacts [7]. The primary method

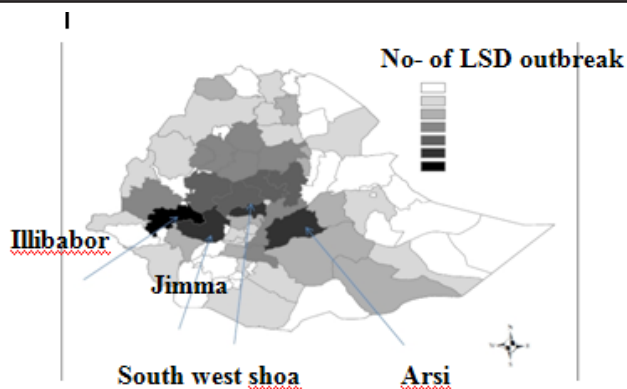


Figure 6: A map depicting the spread of lumpy skin disease outbreaks in Ethiopia between 2007 and 2011. Source: [28].

for controlling LSD in Ethiopia is through ring vaccination conducted at the onset of an outbreak. The National Veterinary Institute (NVI) in Ethiopia produces both Kenyan SGPV and Neethling strain vaccines, with the former being widely used for all cattle, sheep, and goats. The vaccine provides protection for a minimum of three years [4].

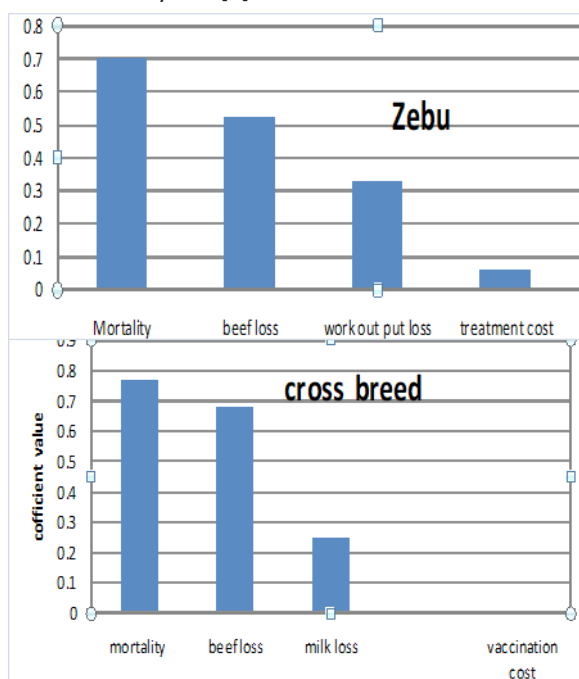


Figure 7: A sensitivity analysis conducted to examine the financial cost estimates for indigenous zebu cattle and Holstein Friesian (HF) or crossbred cattle using regression coefficients. Source: [4].

Economic importance

Lumpy skin disease is a significant and economically damaging illness in Africa and the Middle East that leads to substantial decreases in cattle production. The disease’s economic impact is mainly attributed to its high morbidity rate rather than its mortality [3]. This impact can be broadly categorized into direct and indirect losses. Direct losses encompass visible effects such as animal death, illness, or stunting due to the disease or control measures [9]. Conversely, invisible losses include less immediate impacts like reduced productivity and changes in herd fertility, leading to a higher proportion of animals being used for breeding rather than production. In countries with limited resources, the slaughter of infected and in-contact animals is often viewed as a loss of valuable food source, affecting stakeholders such as farmer [9].

Indirect losses also play a crucial role, including the forgone

revenues due to bans on international trade of livestock, reduced consumer confidence, and negative effects on other sectors of the economy. Large outbreaks can disrupt the dynamics of supply and demand for animals and animal products, with impacts extending beyond individual farms. Additionally, the costs associated with mitigation and control efforts, such as drugs, vaccines, surveillance, and labor, should be considered. These costs may also impact taxpayers due to the need for additional resources to implement control programs [9].

The consequences of lumpy skin disease are far-reaching and include retarded genetic improvement, reduced productivity in cattle industries, bans on international trade of livestock, and expenses related to annual mortality, treatment, and vaccination [10].

The disease causes lesions in the skin, subcutaneous tissue, and muscles of limbs, leading to severe inflammation and reduced mobility due to secondary infection of lesions. These factors result in significant economic losses and decreased productivity [23].

Based on [4], the annual financial impact of a Lumpy Skin Disease (LSD) outbreak in Ethiopia is determined by adding up the annual production losses from illness and death, along with the expenses for treatment and vaccination. The treatment cost accounts for the money spent by farmers on medication.

The total financial costs (C) can be expressed as $C = Md + (B + M + Wop) + V + T$, where M represents the losses in milk production, B denotes the losses in beef production, Wop signifies the losses in work output, Md stands for mortality losses, V indicates vaccination costs, and T represents treatment costs.

The occurrence of LSD disrupts the normal functioning of livestock herds, resulting in a decrease in surplus due to mortality or a reduction in the number of animals available for sale in affected herds because of prolonged illness that can lead to reduced weight gain. The assessment of the loss of draft power depends on when an ox falls ill during the crop season and the corresponding need for draft power at that specific time. The diminished work output of draft oxen as a result of LSD is a significant setback for the mixed crop-livestock farming system. The illness of draft oxen leads to decreased crop production through reduced cultivation and lower yields due to inefficient land preparation and timing [4].

According to [4], the financial impact of disease on local zebu cattle compared to HF/crossbreds indicates that HF/crossbreds experience significantly greater production losses across various parameters when compared to local zebu cattle. This financial impact is directly related to the incidence of the disease in each breed. Infected herds have reported milk production losses of up to 50% per lactation, particularly affecting high-producing exotic breeds, highlighting the significance of LSD infection.

Furthermore, feedlot owners incurred substantial economic losses due to the extra feed required to aid sick animals during their recovery, as well as the extended fattening period. Animals that recuperated were no longer suitable for export and were consequently sold at local markets for a lower price. The survey also revealed that recovered animals produced less milk and experienced a decline in draught power [28].

Overall, LSD is considered a disease with significant economic implications due to its impact on food security through protein loss, reduced animal production output, increased production

costs for disease control, disruption of livestock and product trade, reduced milk yield, weight loss, abortion, infertility in cows, mastitis, infertility in lactating cows, infertility in bulls. Permanent damage to the skin and hide significantly affects the leather industry. It also leads to a ban on international livestock trade and results in prolonged economic losses when it becomes endemic, causing serious stock loss [21,7].

Conclusions and recommendations

Lumpy Skin Disease (LSD) is a viral illness in domestic cattle caused by viruses from the Capripoxvirus genus and is considered to be one of the most economically impactful transboundary diseases. Its effects on animals are significant, leading to persistent weakness, reduced milk production and weight, damaged hides, abortion, and death. Presently, LSD has become established as an endemic disease in the majority of African and Middle Eastern countries. Transmission of LSDV among cattle occurs through mechanical vectors such as blood-feeding arthropods. The significance of various mechanical vectors in the spread of LSDV is expected to differ across different geographical regions, influenced by factors such as environmental conditions, temperature, humidity, and vector abundance. LSD tends to be more prevalent during the wet season, typically at the end of summer and the start of autumn. Controlling LSD involves strategies such as vaccination, restricting animal movement, and eliminating infected and exposed animals.

Based on the above findings, the following recommendations are proposed:

Enhanced measures to control illegal livestock and animal product movements should be considered.

Government and non-governmental organizations should facilitate initiatives for raising awareness and providing training to farmers and veterinary staff to recognize the disease in field conditions.

If LSD enters a disease-free country, rapid detection and prompt culling of infected herds and carcasses, along with ring vaccination, should be considered.

To effectively control LSDV in endemic countries, it is crucial to gain a comprehensive understanding of the ecology of various blood-feeding and biting arthropod species in cattle farming environments.

- Suspected LSD-infected animals should be isolated, and the farm should be quarantined until a definitive diagnosis is determined.

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